A Dissertation On

EFFECTIVENESS OF COLD HIP BATH ON PATIENTS WITH EARLY DIABETES MELLITUS

Submitted by

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I, **Dr. K. Sivakumaran** solemnly declare that this dissertation entitled **"EFFECTIVENESS OF COLD HIP BATH ON PATIENTS WITH EARLY DIABETES MELLITUS"** is a bonafide and genuine research work carried out by me at Government Yoga and Naturopathy Medical College and Hospital, Chennai from July 2016 - June 2017 under the guidance and supervision of

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The Institutional Ethical Committee of Government Yoga & Naturopathy Medical College and Hospital, Chennai reviewed and discussed the application for approval of "EFFECTIVENESS OF COLD HIP BATH ON PATIENTS WITH EARLY DIABETES MELLITUS", project work submitted by Dr. K. Sivakumaran, 2nd year M.D.Naturopathy, Post graduate, Government Yoga and Naturopathy Medical College and Hospital, Chennai.

The proposal is APPROVED.

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List of Abbreviations

FBS – Fasting blood sugar

PPBS – Post Pranidal Blood Sugar

BMI – Body Mass Index

IGT – Impaired Glucose Tolerance

IFG – Impaired Fasting Glucose

TFC7L2 – Transcription Factor 7 like 2

SNP - Single-Nucleotide Polymorphism

MODY – Maturity onset Diabetes of Youth

HNF- Hepatocyte nuclear factors

IPF- Insulin promoter factor

OPD – Out Patient Department

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Introduction:

Diabetes mellitus type II is a group of heterogeneous disorders characterized by variable degrees of insulin resistance, variable insulin secretion, and increased glucose production. Recent studies on diabetes prevalence predict that an estimated 366 million people will have diabetes by 2030. India has about 33 million subjects with diabetes in urban population.

The prevalence rate in India is 2.4%in rural areas and 11.9% in urban areas associated with high impaired glucose tolerance in urban population

Diabetes can lead to systemic complications like neuropathy, nephropathy, retinopathy, vascular complications.

The costs of therapy in conventional medicine are growing high and the disease burden is constantly increasing currently it is about 50%to 75% of household costs

This can be effectively reduced by the use of lifestyle changes and simple therapies which have been said to have marked effect to blood sugar levels.

There are several practices that help in managing diabetes mellitus, but there is necessity for further rigorous scientific evaluations about the efficacy, safety, mechanisms of action. Hence this study aims to study and understand the effect of cold hip bath in managing diabetes mellitus type II and reducing blood sugar levels.

Review of literature:

General Information on Diabetes mellitus:

Definition:

Type II diabetes Mellitus is a subclass of diabetes mellitus that is not insulin responsive or dependent; characterized initially by insulin resistance and hyper insulinemia and eventually by glucose intolerance, hyperglycemia, and overt diabetes; type ii diabetes mellitus is no longer considered a disease exclusively found in

adults; patients seldom develop ketosis but often exhibit obesity.[1]

Etiology:

The etiology of type 2 diabetes mellitus appears to involve complex interactions between environmental and genetic factors. Presumably, the disease develops when a diabetogenic lifestyle. [2] (ie, excessive caloric intake,

inadequate caloric expenditure, obesity) is superimposed on a susceptible genotype.

The body mass index (BMI) at which excess weight increases risk for diabetes varies with different racial groups. For example, compared with persons of European ancestry, persons of Asian ancestry are at increased risk for diabetes at lower levels of overweight. Hypertension and pre hypertension are associated with a greater risk of developing diabetes in whites than in African Americans.

In addition, an inutero environment resulting in low birth weight may predispose some individuals to develop type 2 diabetes mellitus. Infant weight velocity has a small, indirect effect on adult insulin resistance, and this is primarily mediated through its effect on BMI and waist circumference.

About 90% of patients who develop type 2 diabetes mellitus are obese. However, a large, population-based, prospective study has shown that an energy-dense diet may be a risk

factor for the development of diabetes that is independent of baseline obesity.

Some studies suggest that environmental pollutants may play a role in the development and progression of type 2 diabetes mellitus. A structured and planned platform is needed to fully explore the diabetes-inducing potential of environmental pollutants.[3]

Secondary diabetes may occur in patients taking glucocorticoids or when patients have conditions that antagonize the actions of insulin (eg, Cushing syndrome, acromegaly, pheochromocytoma).

Classification of Diabetes:[4]

I. Type 1 diabetes
(beta cell destruction, usually leading to absolute insulin deficiency)
A. Immune-mediated B. Idiopathic

II. Type 2 diabetes
(may range from predominantly insulin resistance with relative insulin deficiency to a predominantly secretory defect with insulin resistance)

III. Other specific types

IV. Gestational diabetes mellitus

Genetic defects of betacell function
- Chromosome 20, HNF-4alpha (MODY1)
- Chromosome 7, glucokinase (MODY2)
- Chromosome 12, HNF-1alpha (MODY3)
- Chromosome 13, IPF-1 (MODY4)
- Chromosome 17, HNF-1beta (MODY5)
- Chromosome 2, NeuroD1 (MODY6)
- Chromosome 2, KLF11 (MODY7)
- Chromosome 9, CEL (MODY8)
- Chromosome 7, PAX4 (MODY9)
- Chromosome 11, INS (MODY10)
- Chromosome 8, BLK (MODY11)
- Mitochondrial DNA
- Permanent neonatal diabetes
- Transient neonatal diabetes
- Others

Genetic defects in insulin action
- Leprechaunism
- Lipoatrophic diabetes
- Rabson-Mendenhall syndrome
- Type A insulin resistance
- Others

Diseases of the exocrine pancreas
- Cystic fibrosis
- Fibrocalculouspancreatopathy
- Hemochromatosis
- Neoplasia
- Pancreatitis
- Trauma/pancreatectomy
- Others

Endocrinopathies
- Acromegaly
- Aldosteronoma
- Cushing's syndrome
- Glucagonoma
- Hyperthyroidism
- Pheochromocytoma
- Somatostatinoma
- Others

Drug- or chemical-induced
- Alpha-interferon
- Atypical antipsychotics
- Beta-adrenergic agonists
- Diazoxide
- Dilantin
- Glucocorticoids
- Highly Active Antiretroviral Therapy (HAART)
- HMG CoA reductase inhibitors (statins)
- Nicotinic acid
- Pentamidine
- Thiazides
- Thyroid hormone
- Vacor (rodenticide)
- Others

Infections
- Congenital rubella
- Cytomegalovirus
- Others

Uncommon forms of immune-mediated diabetes
- Anti-insulin receptor antibodies
- "Stiff-man" syndrome
- Others

Other genetic syndromes sometimes associated with diabetes
- Down syndrome
- Friedreich ataxia
- Huntington chorea
- Klinefelter syndrome
- Laurence-Moon-Bardet-Biedl syndrome
- Myotonic dystrophy
- Porphyria
- Prader-Willi syndrome
- Turner syndrome
- Wolfram syndrome
- Others

Major risk factors:

The major risk factors for type 2 diabetes mellitus are the following:[5]

Age greater than 45 years (though, as noted above, type 2 diabetes mellitus is occurring with increasing frequency in young individuals)

Weight greater than 120% of desirable body weight

Family history of type 2 diabetes in a first-degree relative (eg, parent or sibling)

Hispanic, Native American, African American, Asian American, or Pacific Islander descent

History of previous impaired glucose tolerance (IGT) or impaired fasting glucose (IFG)

Hypertension (>140/90 mm Hg) or dyslipidemia (HDL cholesterol level < 40 mg/dL or triglyceride level >150 mg/dL)

History of gestational diabetes mellitus or of delivering a baby with a birth weight of over 9 lb

Polycystic ovarian syndrome (which results in insulin resistance)

Genetic influences:

The genetics of type 2 diabetes are complex and not completely understood. Evidence supports the involvement of multiple genes in pancreatic beta-cell failure and insulin resistance.

Genome-wide association studies have identified dozens of common genetic variants associated with increased risk for type 2 diabetes. Of the variants thus far discovered, the one with the strongest effect on susceptibility is the transcription factor 7–like 2 (TCF7L2) gene.[6]

Identified genetic variants account for only about 10% of the heritable component of most type 2 diabetes. An international research consortium found that use of a 40-SNP genetic risk score improves the ability to make an

approximate 8-year risk prediction for diabetes beyond that which is achievable when only common clinical diabetes risk factors are used. Moreover, the predictive ability is better in younger persons (in whom early preventive strategies could delay diabetes onset) than in those older than 50 years.

Some forms of diabetes have a clear association with genetic defects. The syndrome historically known as maturity onset diabetes of youth (MODY), which is now understood to be a variety of defects in beta-cell function, accounts for 2-5% of individuals with type 2 diabetes who present at a young age and have mild disease. The trait is autosomal dominant and can be screened for through commercial laboratories.[7],[8]

To date, 11 MODY subtypes have been identified, involving mutations in the following genes:

- HNF-4-alpha
- Glucokinase gene
- HNF-1-alpha

- IPF-1
- HNF-1-beta
- NEUROD1
- KLF11
- CEL
- PAX4
- INS
- BLK

Most of the MODY subtypes are associated with diabetes only; however, MODY type 5 is known to be associated with renal cysts, and MODY type 8 is associated with exocrine pancreatic dysfunction.[9]

A number of variants in mitochondrial deoxyribonucleic acid (DNA) have been proposed as an etiologic factor for a small percentage of patients with type 2 diabetes. Two specific point mutations and some deletions and duplications in the mitochondrial genome can cause type 2 diabetes and sensorineural hearing loss.

Diabetes can also be a finding in more severe mitochondrial disorders such as Kearns-Sayre syndrome and mitochondrial encephalomyopathy, lactic acidosis, and stroke like episode (MELAS).[10] Mitochondrial forms of diabetes mellitus should be considered when diabetes occurs in conjunction with hearing loss, myopathy, seizure disorder, stroke like episodes, retinitis pigmentosa, external ophthalmoplegia, or cataracts. These findings are of particular significance if there is evidence of maternal inheritance.

A meta-analysis of two studies indicated that a genetically associated low birth weight increases an individual's risk for developing type 2 diabetes. The report found that for each one-point increase in an individual's genetic risk score for low birth weight, the type 2 diabetes risk rose by 6%.[11]

Pathophysiology of Diabetes Mellitus:

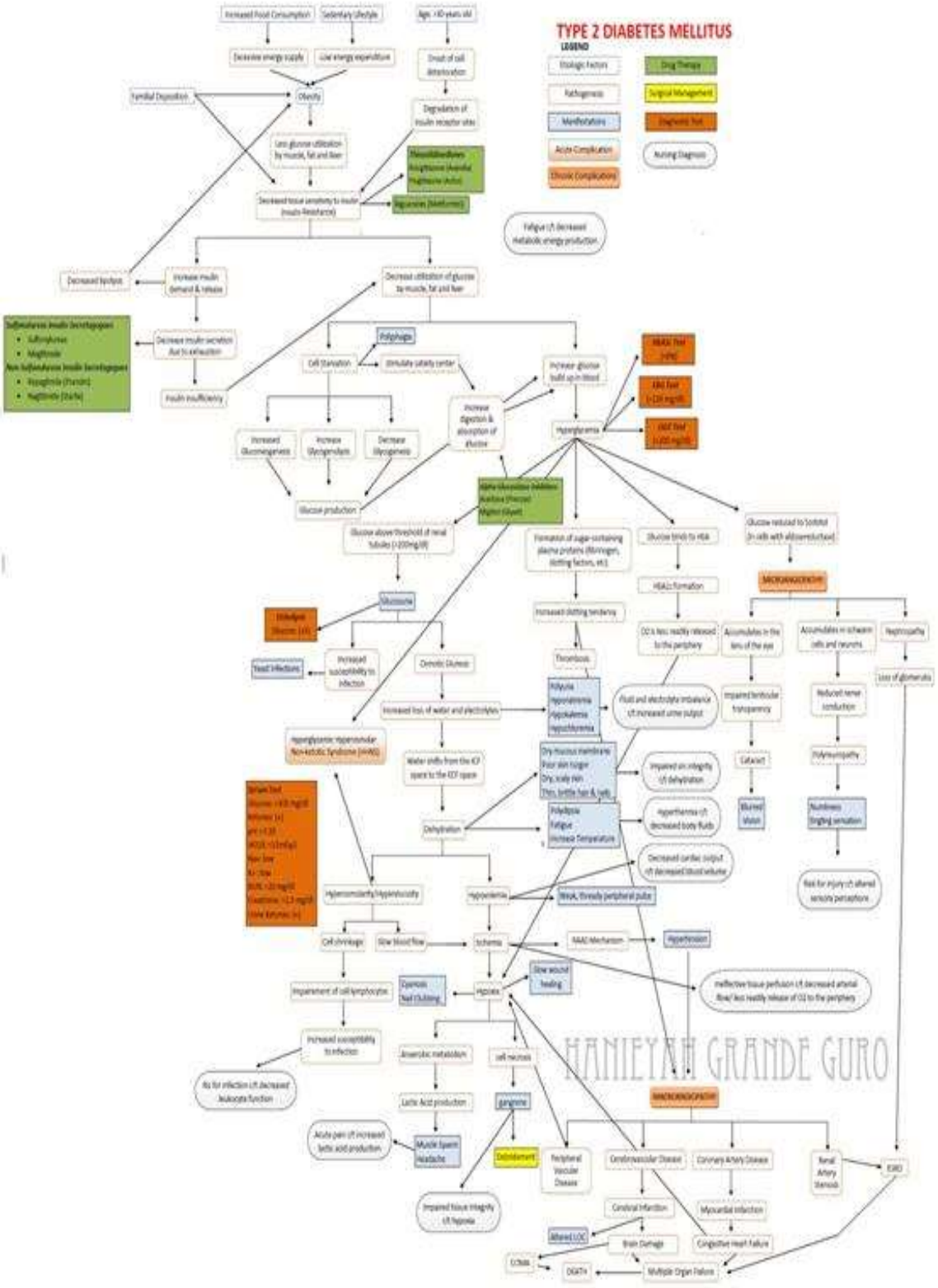
In type 2 diabetes these mechanisms break down, with the consequence that the two main pathological defects in type 2 diabetes are impaired insulin secretion through a dysfunction of the pancreatic β-cell, and impaired insulin action through insulin resistance. [12] In situations where resistance to insulin predominates, the mass of β-cells undergoes a transformation capable of increasing the insulin supply and compensating for the excessive and anomalous demand. In absolute terms, the plasma insulin concentration (both fasting and meal stimulated) usually is increased, although "relative" to the severity of insulin resistance, the plasma insulin concentration is insufficient to maintain normal glucose homeostasis. Keeping in mind the intimate relationship between the secretion of insulin and the sensitivity of hormone action in the complicated control of glucose homeostasis, it is practically impossible to separate the contribution of each to the etiopathogenesis of DM2 [13].

Insulin resistance and hyperinsulinemia eventually lead to impaired glucose tolerance. Except for maturity onset diabetes of the young (MODY), the mode of inheritance for type 2 diabetes mellitus is unclear. MODY, inherited as an autosomal dominant trait, may result from mutations in glucokinase gene on chromosome 7p. MODY is defined as hyperglycemia diagnosed before the age of twenty-five years and treatable for over five years without insulin in cases where islet cell antibodies (ICA) are negative.

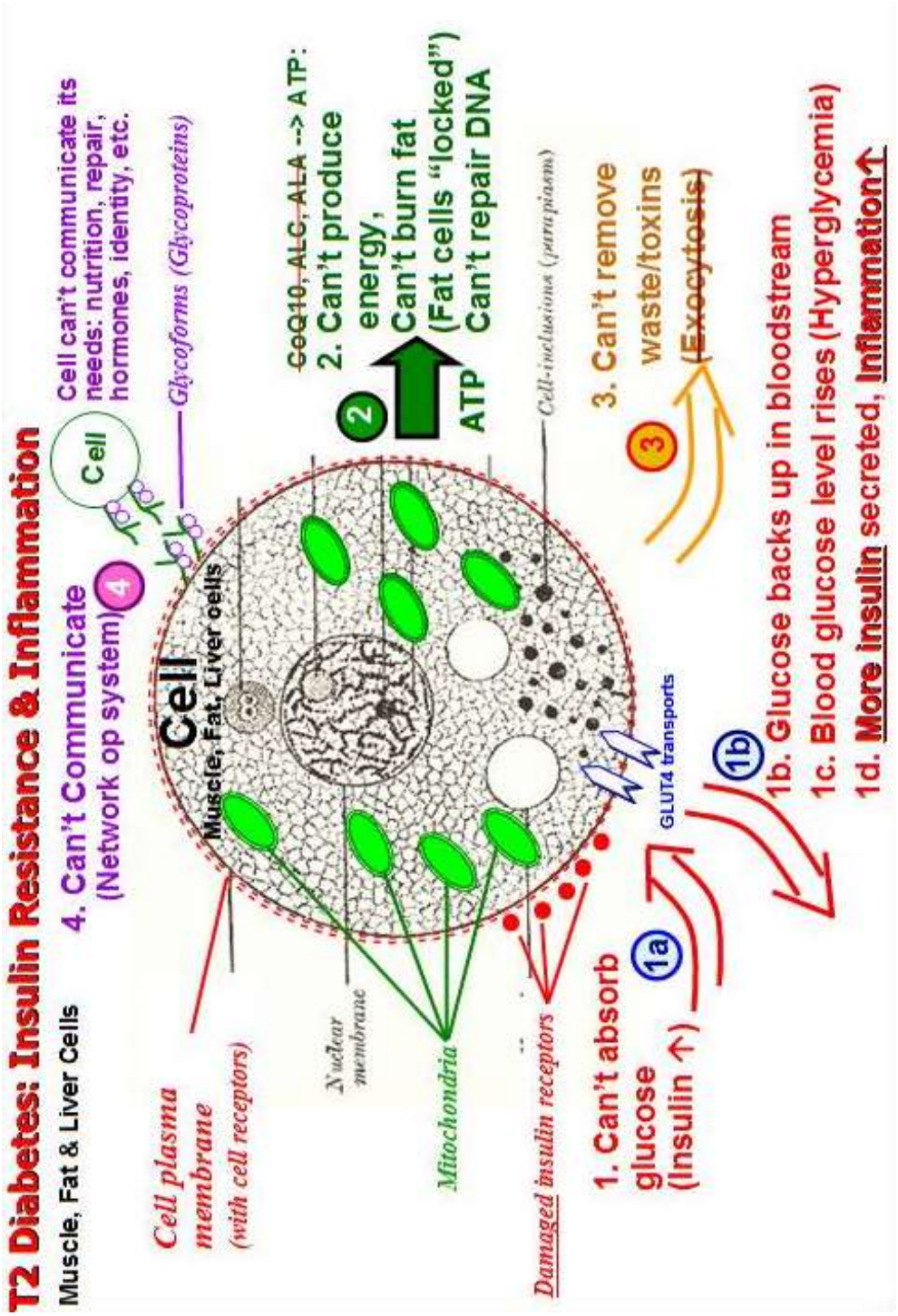
Insulin resistance

The primary events are believed to be an initial deficit in insulin secretion and in many patients relative insulin deficiency in association with peripheral insulin resistance [14]. Resistance to the action of insulin will result in impaired insulin mediated glucose uptake in the periphery (by muscle and fat), incomplete suppression of hepatic glucose output and impaired triglyceride uptake by fat. To overcome the insulin resistance, islet cells will increase the amount of insulin secreted. Endogenous glucose production is

accelerated in patients with type 2 diabetes or impaired fasting glucose. Because this increase occurs in the presence of hyper insulinemia, at least in the early and intermediate disease stages, hepatic insulin resistance is the driving force of hyperglycemia of type 2 diabetes


TYPE 2 DIABETES MELLITUS
LEGEND
Etiologic Factors
Pathogenesis
Manifestations
Acute Complication
Chronic Complications
Drug Therapy
Surgical Management
Diagnostic Test
Nursing Diagnosis
Increased Food Consumption
Sedentary Lifestyle
Age: >40 years old
Decrease energy supply
Low energy expenditure
Onset of cell deterioration
Familial Disposition
Obesity
Degradation of insulin receptor sites
Less glucose utilization by muscle, fat and liver
Decreased tissue sensitivity to insulin (Insulin Resistance)
Fatigue r/t decreased metabolic energy production
Decreased lipolysis
Increase insulin demand & release
Decrease utilization of glucose by muscle, fat and liver
Sulfonylureas insulin Secretagogues
Sulfonylureas
Meglitinide
Non-Sulfonylureas Insulin Secretagogues
Repaglinide (Prandin)
Nateglinide (Starlix)
Decrease insulin secretion due to exhaustion
Insulin insufficiency
Polyphagia
Cell Starvation
Stimulate satiety center
Increase glucose build up in blood
Increase digestion & absorption of glucose
Hyperglycemia
Increased Gluconeogenesis
Increase Glycogenolysis
Decrease Glycogenesis
Glucose production
Glucose above threshold of renal tubules (>200mg/dl)
Glucosuria
Diuresis (+)
Yeast Infections
Increased susceptibility to infection
Osmotic Diuresis
Increased loss of water and electrolytes
Hyperglycemic Hyperosmolar Non-Ketotic Syndrome (HHNS)
Water shifts from the ICF space to the ECF space
Dehydration
Hyperosmolarity/Hyperviscosity
Cell shrinkage
Slow blood flow
Impairment of cell lymphocyte
Increased susceptibility to infection
Rx for infection r/t decreased leukocyte function
Anaerobic metabolism
Lactic Acid production
Acute pain r/t increased lactic acid production
Muscle Spasm Headache
Impaired tissue integrity r/t hypoxia
Glucose above threshold of renal tubules (>200mg/dl)
Formation of sugar-containing plasma proteins (fibrinogen, clotting factors, etc)
Glucose binds to HBA
HBA1c formation
Increased clotting tendency
Thrombosis
Polyuria Hypovolemia Hypokalemia Hyponatremia
Increased loss of water and electrolytes
Dry mucous membrane Poor skin turgor Dry, scaly skin Thin, brittle hair & nails
Polydipsia Fatigue Increase Temperature
Hypovolemia
Weak, thready peripheral pulse
Ischemia
Hypoxia
Slow wound healing
Gangrene
Delayed wound healing
Peripheral Vascular Disease
Cerebrovascular Disease
Coronary Artery Disease
Renal Artery Stenosis
ESRD
Cerebral Infarction
Myocardial Infarction
Altered LOC
Brain Damage
Congestive Heart Failure
Coma
DEATH
Multiple Organ Failure
Glucose reduced to tolerated (in cells with dysfunction)
MICROANGIOPATHY
Accumulates in the lens of the eye
Accumulates in schwann cells and neurons
Nephropathy
Loss of glomerulus
Fluid and electrolyte imbalance r/t increased urine output
Impaired lenticular transparency
Cataract
Blurred Vision
Reduced nerve conduction
Polyneuropathy
Numbness tingling sensation
Risk for injury r/t altered sensory perception
Impaired skin integrity r/t dehydration
Hypothermia r/t decreased body fluids
Decreased cardiac output r/t decreased blood volume
Hypertension
Ineffective tissue perfusion r/t decreased arterial flow/ less readily release of O2 to the periphery
MACROANGIOPATHY
HANIEYAH GRANDE GURO

Effectiv


T2 Diabetes: Insulin Resistance & Inflammation
Muscle, Fat & Liver Cells
4. Can't Communicate
(Network op system)
4
Cell can't communicate its needs: nutrition, repair, hormones, identity, etc.
Cell
Glycoforms (Glycoproteins)
Cell plasma membrane
(with cell receptors)
Cell
Muscle, Fat, Liver cells
Nuclear membrane
Mitochondria
Damaged insulin receptors
Cell-inclusions (paraplasm)
CoQ10, ALC, ALA --> ATP:
2. Can't produce energy,
Can't burn fat
(Fat cells "locked")
Can't repair DNA
2
ATP
3. Can't remove waste/toxins
(Exocytosis)
3
1. Can't absorb glucose
(Insulin ↑)
1a
GLUT4 transports
1b
1b. Glucose backs up in bloodstream
1c. Blood glucose level rises (Hyperglycemia)
1d. More insulin secreted, Inflammation↑

Prevalence:

Type 2 diabetes is increasingly prevalent but also largely preventable.

According to the Centers for Disease Control and Prevention (CDC), type 2 diabetes accounts for about 90 to 95 percent of all diagnosed cases of diabetes in adults.

In general

- Research suggests that 1 out of 3 adults have pre diabetes. Of this group, 9 out of 10 don't know they have it.
- 29.1 million People in the United States have diabetes, but 8.1 million may be undiagnosed and unaware of their condition.
- About 1.4 million new cases of diabetes are diagnosed in United States every year.
- More than one in every 10 adults who are 20 years or older has diabetes. For seniors (65 years and older), that figure rises to more than one in four.

- Cases of diagnosed diabetes cost the United States an estimated $245 billion in 2012. This cost is expected to rise with the increasing diagnoses.

In pregnancy and parenting according to the CDC, 4.6 to 9.2 percent of pregnancies may be affected by gestational diabetes. In up to 10 percent of them, the mother is diagnosed with type 2 diabetes just after the pregnancy. The rest of these women have a 35 to 60 percent chance of developing type 2 diabetes within 10 to 20 years. This risk decreases if the woman leads an active lifestyle and maintains an ideal weight.

A child has a 1 in 7 chance of developing diabetes if one parent was diagnosed before age 50. If the parent was diagnosed after age 50, the child has a 1 in 13 chance. The child's risk may be greater if the mother has diabetes. If both parents have diabetes, the child's risk is about 50 percent.

According to Lancet study,

China, India and USA are among the top three countries with a high number of diabetic populations.

After tightening laws on tobacco and alcohol, experts now want a high tax on sugary drinks as they cause a sugar high

that leads to insulin resistance. Ahead of World Health Day (April 7), the Lancet study (to be published online late tonight) said there is a fourfold rise in the number of diabetics – from 108 million in 1980 to 422 million in 2014 and half of them live in India, China, USA, Brazil and Indonesia.

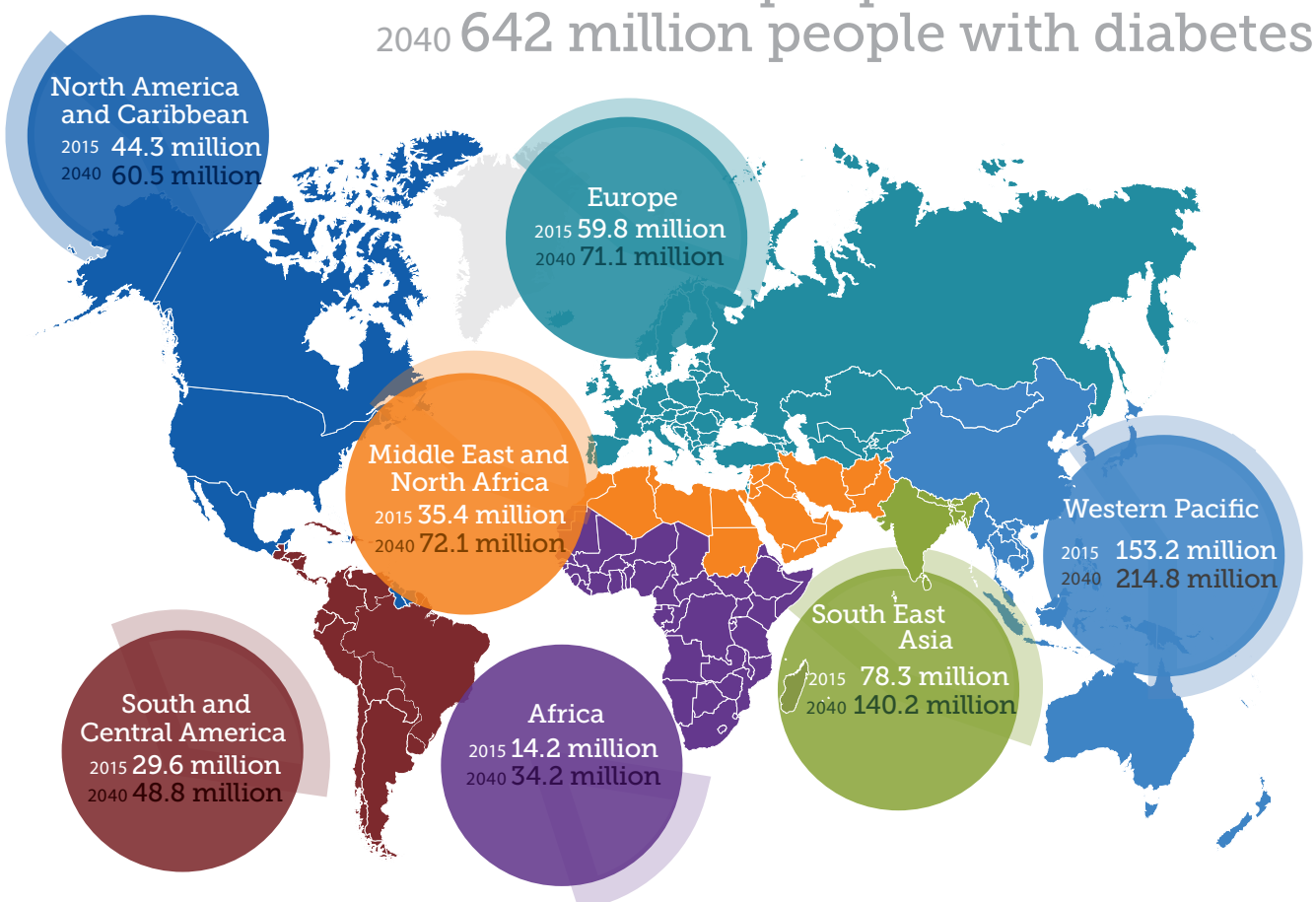
According to the Lancet study, China, India and USA are among the top three countries with a high number of diabetic population. While the numbers climbed from 20.4 million in China in 1980 to 102.9 million in 2014, the rise has been equally dramatic in India from 11.9 million in 1980 to 64.5 million in India. Prevalence of diabetes has more than doubled for men in India and China (3.7 per cent to 9.1 per cent in India and 3.5 per cent to 9.9 per cent in China). It has also increased by 50 per cent among women in China (5.0 per cent to 7.6 per cent) and 80 per cent among women in India (4.6 per cent to 8.3 per cent).[15], [16]

International Diabetes Federation

IDF DIABETES ATLAS

2015
Seventh edition

Worldwide 2015 415 million people with diabetes
2040 642 million people with diabetes



2015

One in 11 adults has diabetes

2040

One in 10 adults will have diabetes

One in two adults with diabetes is undiagnosed

Number of **men** with diabetes

2015 215.2 million
2040 328.4 million

Number of **women** with diabetes

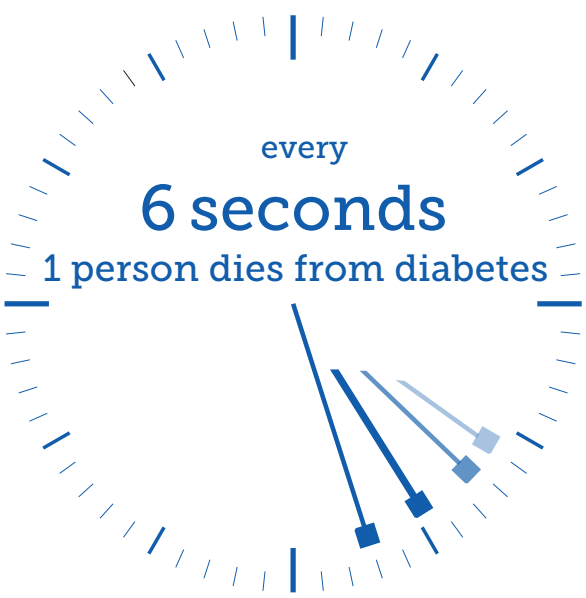
2015 199.5 million
2040 313.3 million

Diabetes in **urban** areas

2015 269.7 million
2040 477.9 million

Diabetes in **rural** areas

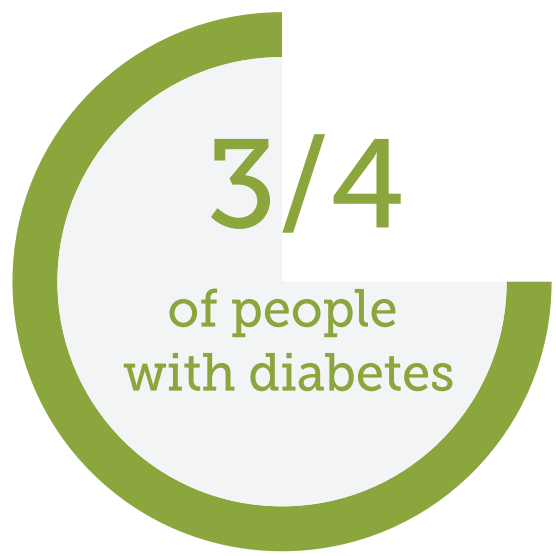
2015 145.1 million
2040 163.9 million



5.0 million deaths in 2015

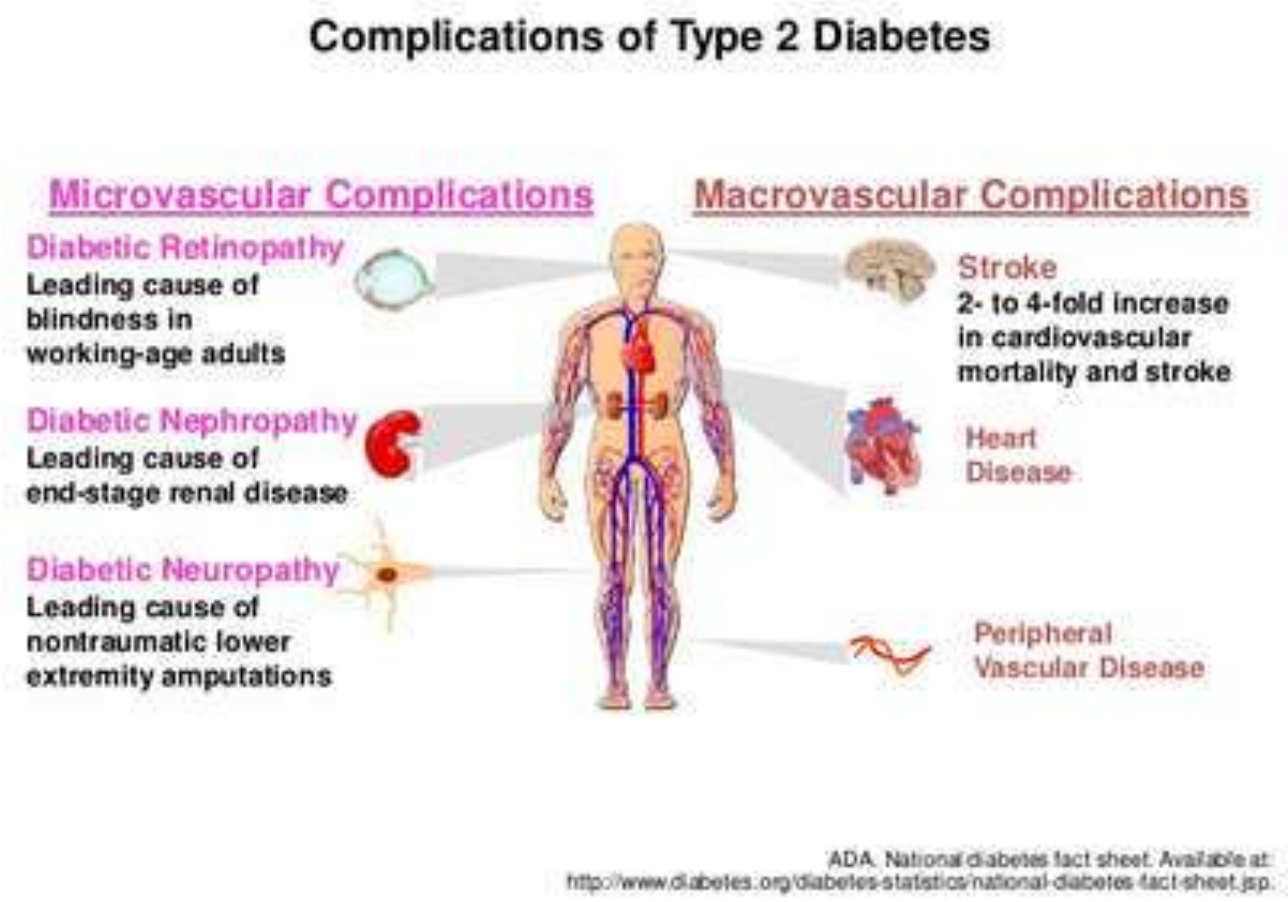
$673 billion

12% of global health expenditure is spent on diabetes



live in low and middle income countries

Complications of Diabetes Mellitus:[17]



The complications of diabetes mellitus

The complications of diabetes mellitus are far less common and less severe in people who have well-controlled blood sugar levels. Acute complications include hypoglycemia and hyperglycemia, diabetic coma and nonketotic hyperosmolar coma. Chronic complications occur due to a mix of microangiopathy, macrovascular disease and immune dysfunction in the form of autoimmune disease or poor immune response, most of which are difficult to manage. Microangipoathy can affect all vital organs, kidneys, heart and brain, as well as eyes, nerves, lungs and locally gums and feet. Macrovascular problems can lead to cardiovascular disease including erectile dysfunction. Female infertility may be due to endocrine dysfunction with impaired signalling on a molecular level.

Other health problems compound the chronic complications of diabetes such assmoking, obesity, high blood pressure,elevated cholesterol levels, and lack of regular exercise which are

accessible to management as they are modifiable. Non-modifiable risk factors of diabetic complications are type of diabetes, age of onset, and genetic factors, both protective and predisposing have been found.[18]

Diabetic ketoacidosis

Diabetic ketoacidosis (DKA) is an acute and dangerous complication that is always a medical emergency and requires prompt medical attention. Low insulin levels cause the liver to turn fatty acid to ketone for fuel (i.e., ketosis); ketone bodies are intermediate substrates in that metabolic sequence. This is normal when periodic, but can become a serious problem if sustained. Elevated levels of ketone bodies in the blood decrease the blood's pH, leading to DKA. On presentation at hospital, the patient in DKA is typically dehydrated, and breathing rapidly and deeply. Abdominal pain is common and may be severe. The level of consciousness is typically normal until late in the process, when lethargy may progress to coma. Ketoacidosis can easily become severe enough to cause hypotension, shock, and death. Urine analysis will reveal significant levels of ketone bodies (which have exceeded their renal threshold blood levels to appear in the urine,

often before other overt symptoms). Prompt, proper treatment usually results in full recovery, though death can result from inadequate or delayed treatment, or from complications (e.g., brain edema). Ketoacidosis is much more common in type 1 diabetes than type 2.

Hyperglycemia hyperosmolar state

Nonketotic hyperosmolar coma (HNS) is an acute complication sharing many symptoms with DKA, but an entirely different origin and different treatment. A person with very high (usually considered to be above 300 mg/dl (16 mmol/L)) blood glucose levels, water is osmotically drawn out of cells into the blood and the kidneys eventually begin to dump glucose into the urine. This results in loss of water and an increase in blood osmolarity. If fluid is not replaced (by mouth or intravenously), the osmotic effect of high glucose levels, combined with the loss of water, will eventually lead to dehydration. The body's cells become progressively dehydrated as water is taken from them and excreted. Electrolyte imbalances are also common and are always dangerous. As with DKA, urgent medical treatment is necessary, commonly

beginning with fluid volume replacement. Lethargy may ultimately progress to a coma, though this is more common in type 2 diabetes than type 1.

Hypoglycemia

Hypoglycemia, or abnormally low blood glucose, is an acute complication of several diabetes treatments. It is rare otherwise, either in diabetic or non-diabetic patients. The patient may become agitated, sweaty, weak, and have many symptoms of sympathetic activation of the autonomic nervous system resulting in feelings akin to dread and immobilized panic. Consciousness can be altered or even lost in extreme cases, leading to coma, seizures, or even brain damage and death. In patients with diabetes, this may be caused by several factors, such as too much or incorrectly timed insulin, too much or incorrectly timed exercise (exercise decreases insulin requirements) or not enough food (specifically glucose containing carbohydrates). The variety of interactions makes cause identification difficult in many instances.

It is more accurate to note that iatrogenic hypoglycemia is typically the result of the interplay of absolute (or relative) insulin excess and compromised glucose counter regulation in type 1 and advanced type 2 diabetes. Decrements in insulin, increments in glucagon, and, absent the latter, increments in epinephrine are the primary glucose counterregulatory factors that normally prevent or (more or less rapidly) correct hypoglycemia. In insulin-deficient diabetes (exogenous) insulin levels do not decrease as glucose levels fall, and the combination of deficient glucagon and epinephrine responses causes defective glucose counter regulation.

Furthermore, reduced sympathoadrenal responses can cause hypoglycemia unawareness. The concept of hypoglycemia-associated autonomic failure (HAAF) in diabetes posits that recent incidents of hypoglycemia causes both defective glucose counter regulation and hypoglycemia unawareness. By shifting glycemic thresholds for the sympathoadrenal (including epinephrine) and the resulting neurogenic responses to lower plasma glucose concentrations, antecedent hypoglycemia leads to a vicious cycle of recurrent hypoglycemia and further impairment of glucose counter regulation. In many cases (but not all), short-term

avoidance of hypoglycemia reverses hypoglycemia unawareness in affected patients, although this is easier in theory than in clinical experience.

In most cases, hypoglycemia is treated with sugary drinks or food. In severe cases, an injection of glucagon (a hormone with effects largely opposite to those of insulin) or an intravenous infusion of dextrose is used for treatment, but usually only if the person is unconscious. In any given incident, glucagon will only work once as it uses stored liver glycogen as a glucose source; in the absence of such stores, glucagon is largely ineffective. In hospitals, intravenous dextrose is often used.

Diabetic coma

Diabetic coma is a medical emergency in which a person with diabetes mellitus is comatose (unconscious) because of one of the acute complications of diabetes:

1. Severe diabetic hypoglycemia
2. Diabetic ketoacidosis advanced enough to result in unconsciousness from a combination of severe hyperglycemia, dehydration and shock, and exhaustion

3. Hyperosmolar nonketotic coma in which extreme hyperglycemia and dehydration alone are sufficient to cause unconsciousness.

Microangiopathy

The damage to small blood vessels leads to a microangiopathy, which can cause one or more of the following:

- Diabetic nephropathy, damage to the kidney which can lead to chronic renal failure, eventually requiring renal dialysis. It is the most common cause of adult kidney failure in the developed world.
- Diabetic neuropathy, abnormal and decreased sensation, usually in a 'glove and stocking' distribution starting with the feet but potentially in other nerves, later often fingers and hands. When combined with damaged blood vessels this can lead to diabetic foot (see below). Other forms of diabetic neuropathy may present as mononeuritis or autonomic neuropathy. Diabetic amyotrophy is muscle weakness due to neuropathy.

- Diabetic retinopathy, growth of friable and poor-quality new blood vessels in the retina as well as macular edema (swelling of the macula), which can lead to severe vision loss or blindness. Retinopathy it the most common cause of blindness among non-elderly adults in the developed world.
- Diabetic encephalopathy is the increased cognitive decline and risk of dementia, including (but not limited to) the Alzheimer's type, observed in diabetes. Various mechanisms are proposed, like alterations to the vascular supply of the brain and the interaction of insulin with the brain itself.
- Diabetic cardiomyopathy, damage to the heart muscle, leading to impaired relaxation and filling of the heart with blood (diastolic dysfunction) and eventually heart failure; this condition can occur independent of damage done to the blood vessels over time from high levels of blood glucose.
- Erectile Dysfunction: Estimates of the prevalence of erectile dysfunction in men with diabetes range from 20 to 85 percent when defined as consistent inability to have an erection firm enough for sexual intercourse. Among men with erectile dysfunction, those with diabetes are likely to have experienced

the problem as much as 10 to 15 years earlier than men without diabetes.

- Periodontal disease (gum disease) is associated with diabetes which may make diabetes more difficult to treat.[14] A number of trials have found improved blood sugar levels in type 2 diabetics who have undergone peridontal treatment.

Macrovascular disease

Macrovascular disease leads to cardiovascular disease, to which accelerated atherosclerosis is a contributor:

- Coronary artery disease, leading to angina or myocardial infarction ("heart attack")
- Diabetic myonecrosis ('muscle wasting')
- Peripheral vascular disease, which contributes to intermittent claudication (exertion-related leg and foot pain) as well as diabetic foot
- Stroke (mainly the ischemic type)
- Carotid artery stenosis does not occur more often in diabetes, and there appears to be a lower prevalence of abdominal aortic

aneurysm. However, diabetes does cause higher morbidity, mortality and operative risks with these conditions.

- Diabetic foot, often due to a combination of sensory neuropathy (numbness or insensitivity) and vascular damage, increases rates of skin ulcers (diabetic foot ulcers) and infection and, in serious cases, necrosis and gangrene. It is why it takes longer for diabetics to heal from leg and foot wounds and why diabetics are prone to leg and foot infections. In the developed worldt is the most common cause of non-traumatic adult amputation, usually of toes and or feet.
- Female infertility is more common in women with diabetes type 1, despite modern treatment, also delayed puberty and menarche, menstrual irregularities (especially oligomenorrhoea),

  mild hyperandrogenism, polycystic ovarian syndrome, fewer live born children and possibly earlier menopause. Animal models indicate that on the molecular level diabetes causes defective leptin, insulin and kiss peptin signaling.

Abnormal immune responses

The immune response is impaired in individuals with diabetes mellitus. Cellular studies have shown that hyperglycemia both reduces the function of immune cells and increases inflammation.

- Respiratory infections such as pneumonia and influenza are more common among individuals with diabetes. Lung function is altered by vascular disease and inflammation, which leads to an increase in susceptibility to respiratory agents. Several studies also show diabetes associated with a worse disease course and slower recovery from respiratory infections.
- Restrictive lung disease is known to be associated with diabetes. Lung restriction in diabetes could result from chronic low-grade tissue inflammation, microangiopathy, and/or accumulation of advanced glycation end products. In fact the presence restrictive lung defect in association with diabetes has been shown even in presence of obstructive lung diseases like asthma and COPD in diabetic patients.
- Lipohypertrophy may be caused by insulin therapy. Repeated insulin injections at the same site, or near to, causes an

accumulation of extra subcutaneous fat and may present as a large lump under the skin. It may be unsightly, mildly painful, and may change the timing or completeness of insulin action.

- Depression was associated with diabetes in a 2010 longitudinal study of 4,263 individuals with type 2 diabetes, followed from 2005-2007. They were found to have a statistically significant association with depression and a high risk of micro and macro-vascular events.[19],[20],[21],[22],[23],[24],[25]

What is Hip Bath:

A Hip bath also known as a sitz bath is one of the most useful types of hydrotherapy. This form of treatment involves covering a person's lower abdomen, buttocks, and upper thighs with enough water. A special type of tub is used for the purpose. The reason for this is so the patient can sit with only the hips and buttocks thus receiving full benefits of the bath. It is a basin made of ceramic or plastic materials. It is used in treating various conditions. The bath is large enough for a person to sit inside it comfortably and its walls are tall enough to keep the hips covered with water.

How is hip bath performed generally?

A hip bath can be taken from 10 to 20 minutes. People who are very thin should not take hip bath more than 10 minutes and the people who are not thin can continue for 20 minutes.

During winter the duration of the hip bath should be reduced by about 25 percent.

Initially, hip bath should be taken only for a minute or two. Gradually, the time can be increased to 10 or 20 minutes. The special tub is filled with water. The level of the water should be adjusted in such a way that it touches a person's navel when they sit in it.

After drinking a glass of warm water, the patient can sit in the tub. The patient's feet should be outside the tub and abdomen thighs must be submerged in the water. One can rest back against the lifted portion of the tub and remain in that position.

Hip Bath Tub

What are the various types of Hip baths?

Hot Hip Bath

The patient should drink one glass of cold water.

- A cold compress should be kept on the head.
- Hot hip bath should begin at 400C.
- The temperature should be gradually increased to 450C.
- Hot hip bath is often taken for 10 minutes at a water temperature of 400C to 450C.
- A cold shower bath should be taken immediately after the hot hip bath.
- It is crucial to prevent the patient from catching a chill after the bath.
- The bath should be stopped immediately, if the patient experiences excessive pain or dizzy.

Cold Hip Bath

- A cold hip bath is a regular treatment in most diseases.
- For a cold hip bath, temperature of the water must be 10C to 18C.
- If the patient feels cold or is very weak, a hot foot immersion should be given in addition to the cold hip bath.
- The period of the bath is generally 10 minutes.
- However, in certain conditions it may differ from one minute to 30 minutes.
- The patient should rub the abdomen vigorously down from the navel and across the body with a fairly coarse wet cloth.
- The legs, feet and upper part of the body should be completely dry during and after the bath.

- Following the cold hip bath, the patient should perform moderate exercise like 'yoga' to warm the body.

- The cold hip bath should not be done in severe inflammations of the pelvic, abdominal region and in painful contractions of the bladder, rectum or vagina.

Neutral Hip Bath

- For a neutral hip bath, temperature of the water should be 320C to 360C.

- Rubbing the abdomen is avoided in this bath.

- This bath is generally taken for 20 minutes to an hour.

Alternate Hip Bath

This bath is also called as "revulsive hip bath".

- For this type of bath, the water temperature in the hot tub should be 400C to 450C.

- In the cold tub, temperature should be 100C to 180C.

- The patient should alternately sit in the hot tub for five minutes and then in the cold tub for three minutes.
- The period of the bath is generally 10 to 20 minutes.
- The head and neck should be kept cold with a cold compress.
- The treatment should terminate with a dash of cold water to the hips.

What are the benefits of hip baths?

Hot hip bath:

A hot hip bath helps to relieve painful menstruation, pain in the pelvic organs, painful urination, inflamed rectum or bladder and painful piles. It also benefits enlarged prostatic gland, painful contractions or spasm of the bladder, sciatica, neuralgia of the ovaries and bladder.

Cold hip bath:

Cold hip bath relieves constipation, indigestion, obesity and helps the eliminative organs to function properly. It is also helpful in uterine problems like irregular menstruation, chronic uterine infections, pelvic inflammation, piles, hepatic congestion, chronic congestion of the prostate gland, seminal weakness, impotency, sterility, uterine and ovarian displacements, dilation of the stomach and colon, diabetes, diarrhea, dysentery, hemorrhage of the bladder and so on.

Neutral hip bath:

The neutral hip bath aid to relieve all acute and sub-acute inflammatory conditions such as acute catarrh of the bladder and urethra and sub-acute inflammations in the uterus, ovaries and tubes. It also relieves neuralgia of the fallopian tubes or testicles, painful spasms of the vagina and prorates of the anus and vulva. In addition, it is a sedative treatment for ‘erotomanis’ in both sexes.

Alternate hip bath:

This bath relieves constant inflammatory conditions of the pelvic viscera, for example, salpingitis, ovaritis, cellulitis and different neuralgias of the genito-urinary organs, sciatica and lumbago.

Cold hip bath – benefits for diabetes

This treatment involves only the hips and abdominal region below the navel.
It is one of the most useful forms of hydrotherapy.
Cold hipbath is very beneficial for diabetes.
It reduces obesity, helps organs of elimination to function properly and relieves constipation and indigestion.

A different type of tub maybe used for cold hip bath.
The tub is filled with enough water (around 4 to 6 liters) so that the hips and lower abdomen up-to the navel are immersed in water when you sit in the tub.
Rest your back on one side of the tub and let legs hang out

on the other side.
A common tub maybe used if the special tub is not available. But you have to place some support under one end to elevate it by 8 to 10 centimeters.

The legs should be adjusted in such a manner, so that no pressure is

exerted on the muscles, ligament and blood vessels of knee region.
Then a coarse wet cloth is used to rub abdomen briskly below navel region.
But remember that during and after bath, the upper portion of the body and legs and feet are kept completely dry. Posted by http://signs-causes-treatment-prevention.blogspot.com Prevention And Detection Of Diseases At An Early Stage When The First Signs To Get The Most Effective Treatment.

The duration of the bath is around 15-20 minutes.
The temperature of water should be maintained between 10 to 18 degrees centigrade.[26]to[48]

OBJECTIVE (S) / AIM:

Aim: Identify the effect of cold hip bath on patients with early type II Diabetes Mellitus

Objective: To understand the ability of Cold hip bath in reducing mean blood sugar levels in individuals with NIDDM

Purpose of the study:

The purpose of the study is to identify and elicit the effectiveness of the hip bath treatment in early diabetes mellitus type 2 patients and its role in reduction of drug intake

The patients who are diagnosed Diabetics with only Diabetes Mellitus are made to undergo Cold Hip bath every day with one break for every 6 days for a period of 6 weeks their FBS and PPBS is regularly monitored twice a week for six weeks.

METHODOLOGY (MATERIALS & METHODS):

Sixty people of age group between 30-50 years recently diagnosed (with in five years of diabetes mellitus diagnosis) with Type 2 Diabetes Mellitus (Non-Insulin dependent) with no known complications, with PPBS less than 300mg/dl are selected from Government Yoga and Naturopathy medical College Hospital O.P.D. are be recruited for the study for the study.

The selected 60 people will be divided into two equally on the basis of randomization – simple randomization (coin toss method) distributed groups. After obtaining informed consent, Pre-interventional FBS & PPBS level of the selected people will be measured and recorded.

The selected people will be given hip bath treatment for the intervention for a period of 6 weeks pre and post FBS and PPBS will be measured and recorded once in two weeks using Glucometer.

Immediately after 6 weeks the post-interventional FBS & PPBS level of the subjects will be measured and recorded.

Subject Selection:

Inclusion Criteria:

1. Patients with NIDDM
2. within 5 to 8 years of diagnosis
3. Age group 30 to 50 years
4. Post Pranidal blood sugar less than 300 mg/dl
5. With no co morbidities / complications
6. People not previously exposed to naturopathy treatements

Exclusion Criteria:

1. Insulin Dependent Diabetes Mellitus
2. Other types of DM (Type 1 DM / GDM)
3. People who already practicing yoga for a month or more.
4. People who presents with high BP, cardio vascular complaints, respiratory complaints, recent surgery, hernia and debilitating diseases.

5. People with other associated complication of DM such as Diabetic Neuropathy, Glaucoma, etc.

Study Design:

The study is a mini randomized control trial. Subjects satisfying the selection criteria were randomized to receive the intervention with 30 subjects in control group and 30 subjects in Intervention group

Statistical Analysis Plan:

The data collected from the values of FBS and PPBS will be analyzed by analysis for change from base line by paired T test for PPBS, analysis for change from base line by Wilcoson signed rank test for FBS, analysis between the groups by Maan whitney test, Multivariate Analysis of variance for relationship.

Results and Interpretation:

TABLE 1: Demographic and other characteristics of study participants

Parameter	Experiment (n=30)	Control (n=30)
Age (years)	39.7 ± 5.6	39.5 ± 4.9
Gender		
Female	16 (53.3)	18 (60.0)
Male	14 (46.7)	12 (40.0)
PPBS (mg/dl)		
Baseline	247.6 ± 21.8	236.2 ± 20.3
Time 1	241.6 ± 21.6	230.6 ± 19.5
Time 2	237.6 ± 21.6	225.5 ± 19.1
Time 3	234.6 ± 21.6	218.8 ± 18.7
Time 4	226.6 ± 21.6	218.5 ± 20.9
Time 5	221.6 ± 21.6	214.3 ± 20.5
Time 6	212.6 ± 21.6	210.1 ± 20.3
Time 7	206.3 ± 21.6	206.0 ± 19.9
Time 8	202.6 ± 21.6	201.9 ± 19.7
Time 9	199.6 ± 21.6	198.5 ± 20.1
Time 10	191.6 ± 21.6	194.5 ± 19.9
Time 11	188.6 ± 21.6	190.6 ± 19.7
Time 12	183.5 ± 21.8	186.8 ± 19.4

FBS (mg/dl)		
Baseline	142.5 ± 17.5	145.3 ± 20.1
Time 1	138.5 ± 17.5	143.4 ± 19.7
Time 2	135.5 ± 17.5	138.4 ± 19.2
Time 3	130.5 ± 17.5	134.7 ± 18.6
Time 4	123.5 ± 17.5	131.2 ± 18.2
Time 5	121.5 ± 17.5	128.6 ± 17.7
Time 6	113.5 ± 17.5	126.0 ± 17.3
Time 7	108.5 ± 17.5	122.9 ± 16.9
Time 8	106.5 ± 17.5	120.6 ± 16.7
Time 9	103.5 ± 17.5	118.0 ± 16.4
Time 10	101.3 ± 16.2	115.7 ± 15.9
Time 11	98.3 ± 15.4	113.5 ± 15.7
Time 12	94.3 ± 15.2	111.1 ± 15.4
Values are presented as Mean ± SD, categorical data presented as n (%).		

TABLE 2: Analysis of change from baseline to post baseline of PPBS using Paired t test for Control group

Parameter	Time Point	n	Mean (SD)	95 % CI for Mean Difference	P-value*
PPBS (mg/dl)	Baseline	30	236.2 (20.3)		
	Time 1	30	230.6 (19.5)		
	Change from baseline to Time 1	30	5.6 (2.3)	(4.8 , 6.5)	< 0.001
	Time 2	30	225.5 (19.1)		
	Change from baseline to Time 2	30	10.7 (2.5)	(9.8 , 11.6)	< 0.001
	Time 3	30	218.8 (18.7)		
	Change from baseline to Time 3	30	17.4 (2.9)	(16.3 , 18.4)	< 0.001

SD : Standard Deviation
95% CI : 95 % Confidence Interval
* P - value is obtained from Parametric Paired t test

Interpretation:

This table indicates that there is an evidence to show the statistically significant reduction in PPBS from baseline to post 1 , 2 and 3 time points in the control group.

TABLE 3: Analysis of change from baseline to post baseline of PPBS using Wilcoxon Signed Rank Test for Control group

Parameter	Time point	n	Median (IQR)	P-value *
PPBS (mg/dl)	Baseline	30	236.0 (228.0 , 252.3)	
	Time 4	30	215.5 (204.8 , 227.3)	
	Change from baseline to Time 4	30	24.0 (23.5 , 26.0)	< 0.001
	Time 5	30	210.5 (200.8 , 222.3)	
	Change from baseline to Time 5	30	28.0 (27.5 , 30.3)	< 0.001
	Time 6	30	206.5 (196.8 , 218.3)	
	Change from baseline to Time 6	30	32.0 (31.5 , 34.3)	< 0.001
	Time 7	30	202.5 (192.8 , 214.3)	
	Change from baseline to Time 7	30	36.0 (35.5 , 38.3)	< 0.001
	Time 8	30	198.5 (188.8 , 210.3)	
	Change from baseline to Time 8	30	40.0 (39.5 , 42.3)	< 0.001
	Time 9	30	195.5 (184.8 , 206.3)	

	Change from baseline to Time 9	30	44.0 (41.8 , 46.0)	< 0.001
	Time 10	30	191.5 (180.8 , 202.3)	
	Change from baseline to Time 10	30	48.0 (46.0 , 50.0)	< 0.001
	Time 11	30	187.5 (176.8 , 198.3)	
	Change from baseline to Time 11	30	52.0 (50.0 , 54.0)	< 0.001
	Time 12	30	183.5 (172.8 , 194.3)	
	Change from baseline to Time 12	30	56.0 (53.0 , 58.0)	< 0.001

IQR is given as (25 th percentile , 75 th percentile)
* P- value is obtained from non-parametric Wilcoxon-Signed Rank Test

Interpretation:

This table indicates that there is an evidence to show the statistically significant reduction in PPBS from baseline to post 4 , 5... and 12 time points in the control group.

TABLE 4: Analysis of change from baseline to post baseline of FBS using Wilcoxon Signed Rank Test for Control group

Parameter	Time point	n	Median (IQR)	P-value *
FBS (mg/dl)	Baseline	30	139.0 (133.0 , 157.8)	
	Time 1	30	138.5 (130.0 , 158.9)	
	Change from baseline to Time 1	30	3.0 (2.8 , 3.1)	< 0.001
IQR is given as (25 th percentile , 75 th percentile)				
* P-value is obtained from non-parametric Wilcoxon-Signed Rank Test				

Interpretation:

This table indicates that there is an evidence to show the statistically significant reduction in FBS from baseline to post 1 time point in the control group.

Parameter	Time Point	N	Mean (SD)	95 % CI for Mean Difference	P-value*
FBS (mg/dl)	Baseline	30	145.3 (20.1)		
	Time 2	30	138.4 (19.2)		
	Change from baseline to Time 2	30	6.9 (1.8)	(6.2 , 7.6)	< 0.001
	Time 3	30	134.7 (18.6)		
	Change from baseline to Time 3	30	10.6 (1.5)	(10.0 , 11.2)	< 0.001
	Time 4	30	131.2 (18.2)		
	Change from baseline to Time 4	30	14.1 (1.9)	(13.4 , 14.8)	< 0.001
	Time 5	30	128.6 (17.7)		
	Change from baseline to Time 5	30	16.7 (2.4)	(15.8 , 17.6)	< 0.001
	Time 6	30	126.0 (17.3)		
	Change from baseline to Time 6	30	19.3 (2.8)	(18.3 , 20.4)	< 0.001
	Time 7	30	122.9 (16.9)		
	Change from baseline to Time 7	30	22.4 (3.1)	(21.2 , 23.6)	< 0.001
	Time 8	30	120.6 (16.7)		
	Change from baseline to Time 8	30	24.8 (3.4)	(23.5 , 26.1)	< 0.001
	Time 9	30	118.0 (16.4)		
	Change from baseline to Time 9	30	27.3 (3.8)	(25.9 , 28.7)	< 0.001
	Time 10	30	115.7 (15.9)		

	Change from baseline to Time 10	30	29.6 (4.1)	(28.1 , 31.2)	< 0.001
	Time 11	30	113.5 (15.7)		
	Change from baseline to Time 11	30	31.9 (4.4)	(30.2 , 33.5)	< 0.001
	Time 12	30	111.1 (15.4)		
	Change from baseline to Time 12	30	34.2 (4.7)	(32.3 , 35.9)	< 0.001
SD : Standard Deviation * P- value is obtained from Parametric Paired t test					

TABLE 5: Analysis of change from baseline to post baseline of FBS using Paired t test for Control group

Interpretation:

This table indicates that there is an evidence to show the statistically significant reduction in FBS from baseline to post 2,3,4... and 12 time points in the control group.

TABLE 6: Analysis of change from baseline to post baseline of PPBS using Paired t test for Experimental group

Parameter	Time Point	n	Mean (SD)	P-value
PPBS (mg/dl)	Baseline	30	247.63 (21.6)	
	Time 1	30	241.63 (21.6)	
	Change from baseline to Time 1	30	6.0 (0)	NA
	Time 2	30	237.63 (21.6)	
	Change from baseline to Time 2	30	10.0 (0)	NA
	Time 3	30	234.63 (21.6)	
	Change from baseline to Time 3	30	13.0 (0)	NA
SD : Standard Deviation NA: Not Applicable .Since, SD is equal for baseline and post baseline.				

Interpretation:

This table indicates that there is an evidence to show the reduction in PPBS from baseline to post 1, 2 and 3 time points in the experimental group.

Note : Technically, we could not get the p value because of the change from baseline to post baseline deviation is zero.

TABLE 7: Analysis of change from baseline to post baseline of PPBS using Wilcoxon Signed Rank Test for Experimental group

Parameter	Time point	n	Median (IQR)	P-value *
PPBS (mg/dl)	Baseline	30	247.0 (233.0 , 264.0)	
	Time 4	30	226.0 (212.0 , 243.0)	
	Change from baseline to Time 4	30	21.0 (21.0 , 21.0)	< 0.001
	Time 5	30	221.0 (207.0 , 238.0)	
	Change from baseline to Time 5	30	26.0 (26.0 , 26.0)	< 0.001
	Time 6	30	212.0 (198.0 , 229.0)	
	Change from baseline to Time 6	30	35.0 (35.0, 35.0)	< 0.001
	Time 7	30	206.0 (192.0 , 223.0)	
	Change from baseline to Time 7	30	41.0 (41.0 , 41.0)	< 0.001
	Time 8	30	202.0 (188.0 , 219.0)	
	Change from baseline to Time	30	45.0 (45.0 , 45.0)	< 0.001

	8			
	Time 9	30	199.0 (185.0 , 216.0)	
	Change from baseline to Time 9	30	48.0 (48.0 , 48.0)	< 0.001
	Time 10	30	191.0 (177.0 , 208.0)	
	Change from baseline to Time 10	30	56.0 (56.0 , 56.0)	< 0.001
	Time 11	30	188.0 (174.0 , 205.0)	
	Change from baseline to Time 11	30	59.0 (59.0 , 59.0)	< 0.001
	Time 12	30	182.5 (166.0 , 199.5)	
	Change from baseline to Time 12	30	64.0 (63.0 , 65.0)	< 0.001
IQR is given as (25 th percentile , 75 th percentile) * P-value is obtained from non-parametric Wilcoxon-Signed Rank Test				

Interpretation:

This table indicates that there is an evidence to show statistically significant the reduction in PPBS from baseline to post 4, 5,6… and 12 time points in the experimental group.

TABLE 8: Analysis of change from baseline to post baseline of FBS using Wilcoxon Signed Rank Test for Experimental group

Parameter	Time point	n	Median (IQR)	P-value *
FBS (mg/dl)	Baseline	30	143.0 (127.0 , 158.3)	
	Time 1	30	139.0 (123.0 , 154.3)	
	Change from baseline to Time 1	30	4.0 (4.0 , 4.0)	< 0.001
IQR is given as (25 th percentile , 75 th percentile) * P-value is obtained from non-Parametric Wilcoxon-Signed Rank Test				

Interpretation:

This table indicates that there is an evidence to show the statistically significant reduction in FBS from baseline to post 1 time point in the experimental group.

TABLE 9: Analysis of change from baseline to post baseline of FBS using Paired t test for Experimental group

Parameter	Time Point	n	Mean (SD)	95 % CI for Mean Difference	P-value*
FBS (mg/dl)	Baseline	30	142.5 (17.5)		
	Time 2	30	135.5 (17.9)		
	Change from baseline to Time 2	30	7.0 (0)	NA	< 0.001
	Time 3	30	130.5 (17.5)		
	Change from baseline to Time 3	30	12.0 (0)	NA	< 0.001
	Time 4	30	123.5 (17.5)		
	Change from baseline to Time 4	30	19.0 (0)	NA	< 0.001
	Time 5	30	121.5 (17.5)		
	Change from baseline to Time 5	30	21.0 (0)	NA	< 0.001
	Time 6	30	113.5 (17.5)		
	Change from baseline to Time 6	30	29.0 (0)	NA	< 0.001

	Time 7	30	108.5 (17.5)		
	Change from baseline to Time 7	30	34.0 (0)	NA	< 0.001
	Time 8	30	106.5 (17.5)		
	Change from baseline to Time 8	30	36.0 (0)	NA	< 0.001
	Time 9	30	103.5 (17.5)		
	Change from baseline to Time 9	30	39.0 (0)	NA	< 0.001
	Time 10	30	101.3 (16.2)		
	Change from baseline to Time 10	30	41.2 (1.9)	(40.4 , 41.9)	< 0.001
	Time 11	30	98.3 (15.4)		
	Change from baseline to Time 11	30	44.2 (2.9)	(43.1 , 45.3)	< 0.001
	Time 12	30	94.3 (15.2)		
	Change from baseline to Time 12	30	48.2 (3.6)	(46.8 , 49.6)	< 0.001

SD : Standard Deviation

NA: Not Applicable. Since, some of the change from baseline to post baseline deviations are zero.

*P-value is obtained from Parametric Paired t test

Interpretation:

This table indicates that there is an evidence to show the statistically significant reduction in FBS from baseline to post 2,3,4 ...12 time points in the experimental group.

TABLE 10: Between groups comparison of change of PPBS using t test

Change from baseline to post baseline	Group	n	Mean	SD	Mean Difference	95% CI of mean difference	P-value *
Change from Baseline to Time 1	Control	30	5.6	2.3	-0.4	(-1.2 , 0.5)	0.39
	Experimental	30	6.0	0			
Change from Baseline to Time 2	Control	30	10.7	2.5	0.7	(-0.3 , 1.6)	0.15
	Experimental	30	10.0	0			
Change from Baseline to Time 3	Control	30	17.4	2.9	4.4	(3.3 , 5.4)	< 0.001
	Experimental	30	13.0	0			

SD: Standard Deviation;

95%CI: 95% Confidence Interval;

*P value is obtained from t test.

Interpretation:

This table indicates that there is an evidence to show the statistically significant difference on PPBS change from baseline to post 3 time point between two groups.

TABLE 11: Between groups comparison of change of PPBS using Mann Whitney Test

Change from Baseline to post baseline	Group	n	Median	IQR	P-value*
Change from Baseline to Time 4	Control	30	24.0	(23.5 , 26.0)	< 0.001
	Experimental	30	21.0	(21.0 , 21.0)	
Change from Baseline to Time 5	Control	30	28.0	(27.5 , 30.3)	< 0.001
	Experimental	30	26.0	(26.0 , 26.0)	
Change from Baseline to Time 6	Control	30	32.0	(31.5 , 34.3)	< 0.001
	Experimental	30	35.0	(35.0 , 35.0)	
Change from Baseline to Time 7	Control	30	36.0	(35.5 , 38.3)	< 0.001
	Experimental	30	41.0	(41.0 , 41.0)	
Change from Baseline to Time 8	Control	30	40.0	(39.5 , 42.3)	< 0.001
	Experimental	30	45.0	(45.0 , 45.0)	
Change from Baseline to Time 9	Control	30	44.0	(41.8 , 46.0)	< 0.001
	Experimental	30	48.0	(48.0 ,	

				48.0)	
Change from Baseline to Time 10	Control	30	48.0	(46.0 , 50.0)	< 0.001
	Experimental	30	56.0	(56.0 , 56.0)	
Change from Baseline to Time 11	Control	30	52.0	(50.0 , 54.0)	< 0.001
	Experimental	30	59.0	(59.0 , 59.0)	
Change from Baseline to Time 12	Control	30	56.0	(53.0 , 58.0)	< 0.001
	Experimental	30	64.0	(63.0 , 65.0)	
IQR is given as (25 th percentile , 75 th percentile) *P value is obtained from non-parametric Mann whitny test.					

Interpretation:

This table indicates that there is an evidence to show the statistically significant difference on PPBS change from baseline to post 4,5,6 …12 time points between two groups.

TABLE 12: Between groups comparison of change of FBS using Mann Whitney Test

Change from baseline to post baseline	Group	n	Median	IQR	P-value*
Change from Baseline to Time 1	Control	30	3.0	(2.8 , 3.1)	< 0.001
	Experimental	30	4.0	(4.0 , 4.0)	
IQR is given as (25 th percentile , 75 th percentile) *P value is obtained from non-parametric Mann whitny test.					

Interpretation:

This table indicates that there is an evidence to show the statistically significant difference on FBS change from baseline to post 1 time point between two groups.

TABLE 13: Between groups comparison of change of FBS using t test

Change from baseline to post baseline	Group	n	Mean	SD	Mean Difference	95% CI of mean difference	P-value *
Change from Baseline to Time 2	Control	30	6.9	1.8	-0.1	(-0.8 , 0.6)	0.77
	Experimental	30	7.0	0			
Change from Baseline to Time 3	Control	30	10.6	1.5	-1.4	(-1.9 , -0.8)	< 0.001
	Experimental	30	12.0	0			
Change from Baseline to Time 4	Control	30	14.1	1.9	-4.9	(-5.6 , -4.2)	< 0.001
	Experimental	30	19.0	0			
Change from Baseline to Time 5	Control	30	16.7	2.4	-4.3	(-5.2 , -3.4)	< 0.001
	Experimental	30	21.0	0			
Change from Baseline to Time 6	Control	30	19.3	2.8	-9.7	(-10.7 , -8.6)	< 0.001
	Experimental	30	29.0	0			

Change from Baseline to Time 7	Control	30	22.4	3.1	-11.6	(-12.8 , -10.4)	< 0.001
	Experimental	30	34.0	0			
Change from Baseline to Time 8	Control	30	24.8	3.4	-11.2	(-12.5 , -9.9)	< 0.001
	Experimental	30	36.0	0			
Change from Baseline to Time 9	Control	30	27.3	3.8	-11.7	(-13.1 , -10.3)	< 0.001
	Experimental	30	39.0	0			
Change from Baseline to Time 10	Control	30	29.6	4.1	-11.5	(-13.2 , -9.8)	< 0.001
	Experimental	30	41.2	1.9			
Change from Baseline to Time 11	Control	30	31.9	4.4	-12.3	(-14.2 , -10.4)	< 0.001
	Experimental	30	44.2	2.9			
Change from Baseline to Time 12	Control	30	34.2	4.7	-13.9	(-16.1 , -11.8)	< 0.001
	Experimental	30	48.2	3.6			
SD : Standard Deviation ; 95%CI : 95%Confidence Interval *P value is obtained from t test.							

Interpretation:

This table indicates that there is an evidence to show the statistically significant difference on FBS change from baseline to post 3,4 ...12 time points between two groups.

TABLE 14: Results of Multivariate Analysis of Variance (MANOVA) for repeated measurements of PPBS

Effect	F[b]	Df	P value	Partial η2*
Time	2906.36	12	<0.001	0.99
Group	0.97	1	0.33	0.02
Time*Group	5748.83	12	<0.001	0.99
b: Exact statistic df : degrees of freedom * Partial Eta Squared				

Interpretation:

From the MANOVA results, the time effect was significant with the p value of <0.001. It means that there is a significant change over time in PPBS (mg/dl). Group effect was not statistically significant (p = 0.33) and the Interaction effect of time and group shows that statistically significant with the p value of <0.001.

Partial η2:
It can be seen that 99% of the variance in PPBS (mg/dl) is explained by the time effect, that 2% of the variance in PPBS (mg/dl) is explained by the overall group effect, and that 99% is explained by the time by group interaction effect.

TABLE 15: Results of Multivariate Analysis of Variance (MANOVA) for repeated measurements of PPBS for the shape of relationship

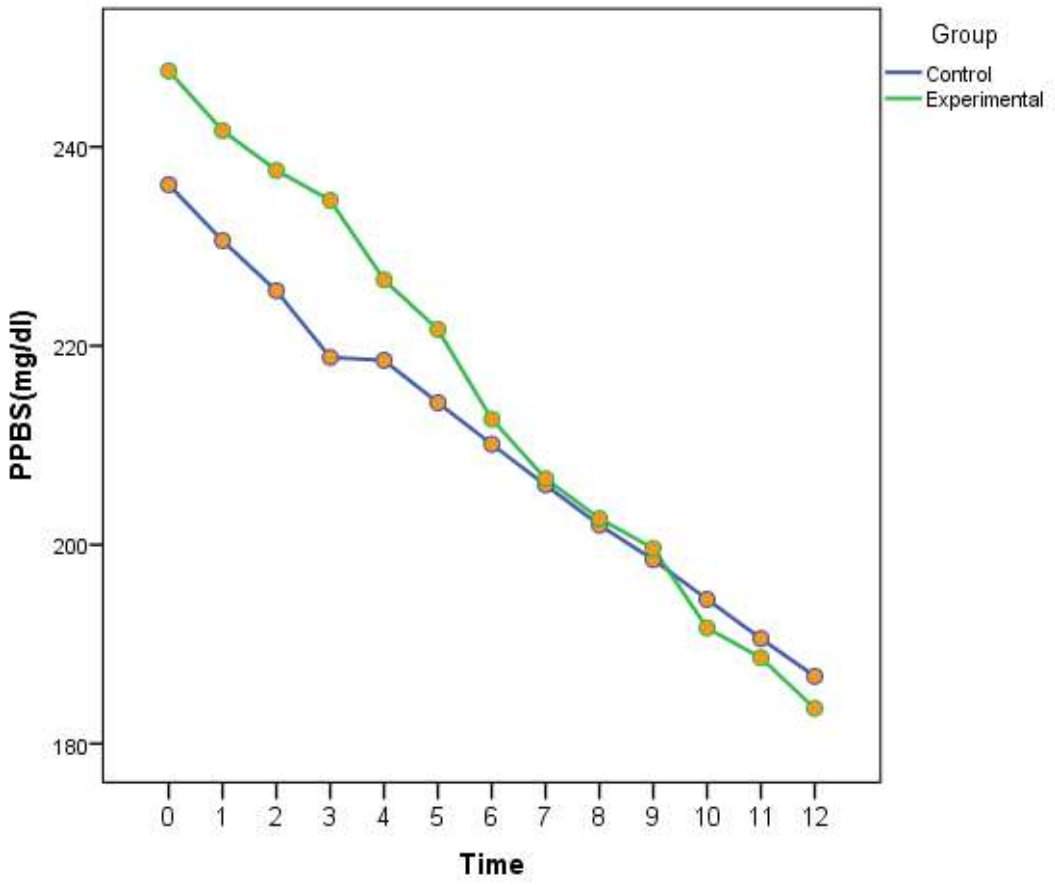
Source	Pattern	Type III Sum of Squares	df	Mean Square	F	P value
Time	Linear	245168.000	1	245168.000	449.007	< 0.001
Time*Group	Cubic	408.218	1	408.218	90.073	< 0.001
df : degrees of freedom						

Interpretation:

Time: The result indicates that there is a statistically significant linear development over the time.

Time*Group: It indicates that there is a statistically significant cubic development over the time.

Figure 1: Development of PPBS over time in the treatment groups



Interpretation:

This figure indicates that overall both group shows the reduction in mean value of PPBS (mg/dl). But especially the experimental group shows the better reduction till the 9 th time point after that the control shows the better. There was an interaction effect.

TABLE 16: Results of Multivariate Analysis of Variance (MANOVA) for repeated measurements of FBS

Effect	F[b]	df	P value	Partial η2*
Time	1412.12	12	<0.001	0.99
Group	5.15	1	0.03	0.08
Time*Group	750.17	12	<0.001	0.99

b: Exact statistic

df : degrees of freedom

* Partial Eta Squared

Interpretation:

From the MANOVA results, the time effect was significant with the p value of <0.001. It means that there is a significant change over time in FBS (mg/dl). Group effect was statistically significant (p = 0.03) and the Interaction effect of time and group shows that statistically significant with the p value of <0.001.

Partial η2:

It can be seen that 99% of the variance in FBS (mg/dl) is explained by the time effect, that 8% of the variance in FBS (mg/dl) is explained by the overall group effect, and that 99% is explained by the time by group interaction effect.

TABLE 17: Results of Multivariate Analysis of Variance (MANOVA) for repeated measurements of FBS for the shape of relationship

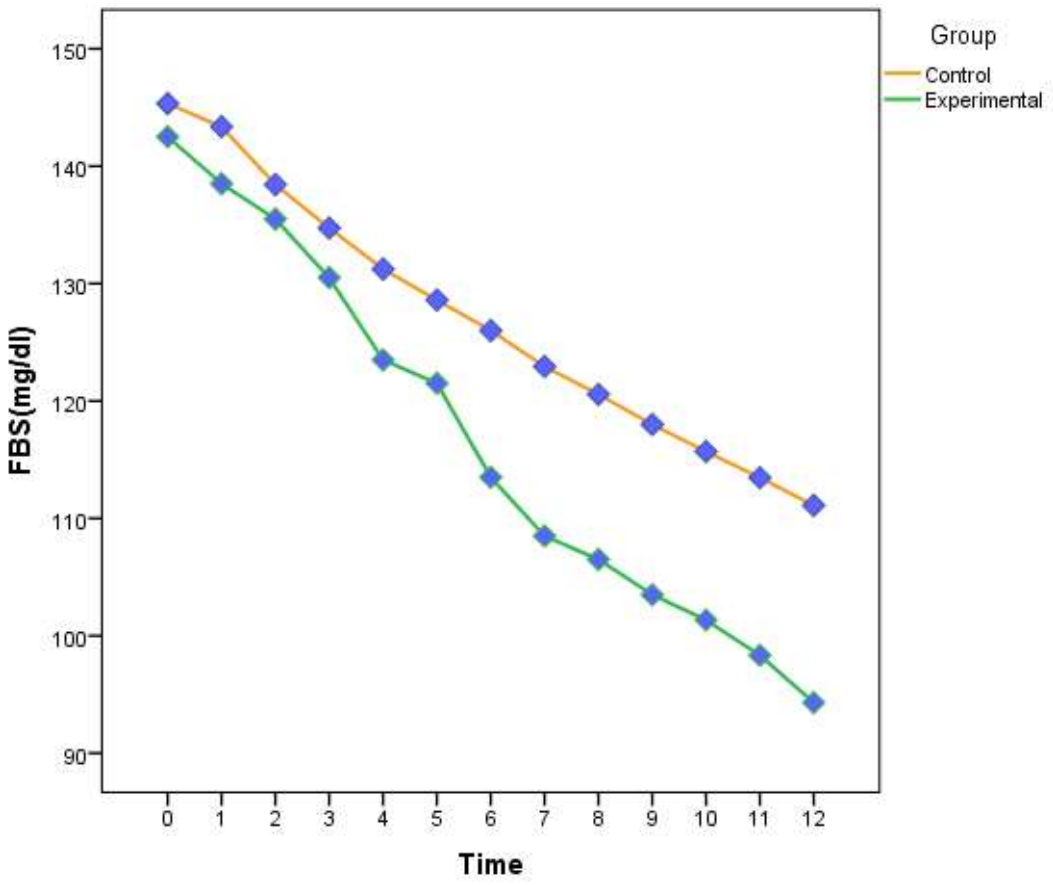
Source	Pattern	Type III Sum of Squares	df	Mean Square	F	P value
Time	Linear	134546.02	1	134546.02	6987.90	<0.001
Time*Group	Order11	51.94	1	51.94	724.93	<0.001
df : degrees of freedom						

Interpretation:

Time: The result indicates that there is a statistically significant linear development over the time.

Time*Group: It indicates that there is a statistically significant order11 pattern development over the time.

Figure 2: Development of FBS over time in the treatment groups



Interpretation:

This figure indicates that overall both group shows the reduction in mean value of FBS (mg/dl). .

Discussion:

The study was done to evaluate the effect of cold hip bath on early type II diabetes mellitus. 36 subjects were recruited for intervention and 34 were selected for control out of which 6 dropped out from intervention and 5 dropped from control, later one additional person was recruited for the control group to maintain 30 in each group

It was found that statistically there is significant difference in the reduction of blood glucose in the use of cold hipbath in addition to the use of conventional medicines when comparing the two groups.

There was a significant change in the blood sugar values in the experimental group participants. But we are not able to conclude that the therapy can work as standalone therapy for type II diabetes mellitus because of the fact that the anti diabetic drugs were not stopped or reduced due to the short nature of the study and significantly smaller sample size. The reduction in blood sugar levels could be due to the increased metabolism as a result of reduction in core temperature where in the body is trying to

reproduce lost temperature where by mimicking exercise. This also reduces the burden on the pancreatic beta cell and insulin resistance.

Limitations of the study:

- The present study has a relatively smaller sample size
- The follow up of the patients is of shorter time, if the time duration would have been greater a clear benefit could have been reported
- Period of the intervention was for a limited period of time.
- Blinding was not possible
- Molecular mechanisms and nervous mechanisms of reduction in blood glucose was not in the scope of the study

Conclusion:

The study was started with the aim of understanding the effect of cold hip bath in reducing diabetes mellitus type II, according to traditional hydrotherapy texts hip bath is an effective method to reduce the blood sugar levels in type II DM, it is also said to increase pancreatic beta cell activity and reduce insulin resistance.

At the end of the study it can be said that the Hip Bath is an effective adjuvant therapy along with conventional medicine to significantly reduce the blood sugar levels, to find out if it can function as a standalone therapy a larger sample size and a longer duration of study would be necessary.

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Appendix i

PROFORMA

Name: Age:

Gender: Male / Female Marital Status:

Religion: Occupation:

Address:

Emergency Contact: Primary Language(s):

Complaints:

History of present Illness:

Previous Illness:

Personal History:

Appetite: Digestion: Sleep:

Bowel: Micturition: Addiction:

Coffee/Tea: with/without sugar Diet :

Family History:

Treatment History:

History of Allergy to any specific drugs/food, if any:

Obstetrics & Gynecology history:

Vital data:

Height:	cms	Weight:	kg
Pulse:	beats/min	Blood Pressure:	mm/Hg
BMI:		Waist Hip ratio:	
Built:		Temperature:	

GENERAL PHYSICAL EXAMINATION:

SYSTEMIC EXAMINATION:

Cardiovascular System:

Respiratory System:

Abdomen:

Nervous System:

Endocrine System:

Genitourinary System:

Locomotor System:

Investigation:

Appendix ii

INFORMATION SHEET

We are conducting a study "Evaluation of the effect of cold hip bath on patients with early Type 2 Diabetes Mellitus" at Government Yoga and Naturopathy Medical College Hospital, Chennai – 106.

The purpose of this study is to evaluate the effectiveness of cold hip bath which may be of use in better management of Diabetes Mellitus.

We need your participation in this study. Here we are assessing the changes in the blood glucose level by measuring the pre and post interventional serum Fasting & PP blood glucose levels were measured at regular intervals.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefit to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator Signature of participant

Date:

INFORMED CONSENT FORM

Title of the study: Effectiveness of cold Hip bath on patients with early Diabetes Mellitus

Name of the Participant:

Name of the Principal Investigator: Dr. K. Sivakumaran.

Name of the Institution: Government Yoga & Naturopathy Medical College, Chennai – 600 106

Documentation of the informed consent

I _____ have read the information in this form (or it has been read to me). I was free to ask any questions and they have been answered. I am over 18 years of age and, exercising my free power of choice, hereby give my consent to be included as a participant in "Effectiveness of cold Hip bath on patients with early Diabetes Mellitus"

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained to me.
3. I have been explained about the nature of the study.
4. I have been explained about my rights and responsibilities by the investigator.
5. I have been informed the investigator of all the treatments I am taking or have taken in the past _____

months including any native (alternative) treatment.

6. I have been advised about the risks associated with my participation in this study.
7. I agree to cooperate with the investigator and I will inform him/her immediately if I suffer unusual symptoms.
8. I have not participated in any research study within the past _____month(s).
9. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital.
10. I am also aware that the investigator may terminate my participation in the study at any time, for any reason, without my consent.
12. I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the sponsors, regulatory authorities, Govt. agencies, and IEC. I understand that they are publicly presented.
13. I have understood that my identity will be kept confidential if my data are publicly presented.
14. I have had my questions answered to my satisfaction.
15. I have decided to be in the research study.

I am aware that if I have any question during this study, I should contact the investigator. By signing this consent form I attest that the information given in this document has been clearly explained to me and understood by me, I will be given a copy of this consent document.

For adult participants:

Name and signature / thumb impression of the participant (or legal representative if participant incompetent)

Name _____ Signature_____

Date_____

Name and Signature of impartial witness (required for illiterate patients):

Name _____ Signature_____

Date_____

Address and contact number of the impartial witness:

Name and Signature of the investigator or his representative obtaining consent:

Name _____ Signature_____

Date_____

INFORMATION TO PARTICIPANTS

Investigator: Dr. K. Sivakumaran

Name of Participant:

Title: Effectiveness of cold Hip bath on patients with early Diabetes Mellitus

You are invited to take part in this research/ study /procedures. The information in this document is meant to help you decide whether or not to take part. Please feel free to ask if you have any queries or concerns. You are being asked to participate in this study being conducted in Government Yoga & Naturopathy Medical College, Chennai – 600 106

What is the Purpose of the Research?

The purpose of the research study is to understand the ability of the hipbath treatment to reduce the blood sugar levels in type II Diabetes Mellitus

The Study Design: Pre Post Matched Control Trial

Study Procedures: Examination of FBS, PPBS

Possible Risks to you: Nil

Possible benefits to you: Possibility to reduce the drug therapy, Maintenance of tight glycemic control, Regular monitoring of blood sugar.

Possible benefits to other people

Confidentiality of the information obtained from you

You have the right to confidentiality regarding the privacy of your medical information (personal details, results of physical examinations, investigations, and your medical history). By signing this document, you will be allowing the research team investigators, other study personnel, sponsors, IEC and any person or agency required by law like the Drug Controller General of India to view your data, if required.

The information from this study, if published in scientific journals or presented at scientific meetings, will not reveal your identity.

How will your decision to not participate in the study affect you?

Your decisions to not to participate in this research study will not affect your medical care or your relationship with investigator or the institution. Your doctor will still take care of you and you will not lose any benefits to which you are entitled.

Can you decide to stop participating in the study once you start?

The participation in this research is purely voluntary and you have the right to withdraw from this study at any time during course of the study without giving any reasons.

However, it is advisable that you talk to the research team prior to stopping the treatment

ஆராய்ச்சி ஒப்புதல் படிவம்

ஆய்வு தலைப்பு:

இடுப்பு குளியல் சிகிச்சையின் மூலம் சர்க்கரை நோயாளிகளின் இரத்த சர்க்கரை அளவில் சிகிச்சைக்கு முன் பின் ஏற்படக்கூடிய மாற்றத்தை ஆராய்ந்து அறியும் ஆய்வு

ஆராய்ச்சியாளர் பெயர்: சிவகுமரன்.கி

ஆராய்ச்சி நடக்கும் இடம் : அரசு யோகா மற்றும் இயற்கை மருத்துவக் கல்லூரி , 106 – சென்னை ,அரும்பாக்கம்

பங்கு பெறுபவரின் பெயர்: *வயது:*

பாலினம்: ஆண் / பெண்

பங்கு பெறுபவரின்அடையாள எண்:

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கமும் முழுமையாக தெளிவாக எனக்கு விளக்கப்பட்டது.எனக்கு விளக்கப்பட்ட தகவல்களை புரிந்து கொண்டு ஆராய்ச்சியில் பங்குபெற எனது சம்மதத்தைத் தெரிவிக்கிறேன்.

நான் இந்த ஆராய்ச்சியில் பங்குபெறவும் அனைத்து தேவையான ஆராய்ச்சிக்கு , பரிசோதனைகளை மேற்கொள்ளவும் முழு சம்மதம் தெரிவிக்கிறேன்.

இந்த ஆராய்ச்சியில் இருந்து நான் எந்த நேரமும் பின்வாங்கலாம் என்றும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் புரிந்து கொண்டேன்.

நான் என்னுடைய சுயநினைவுடனும் முழு ,மனதோடும் இந்த ஆராய்ச்சியில் பங்கேற்க்க சம்மதிக்கிறேன்.

ஆராய்ச்சியாளர் கையொப்பம் *பங்கு பெறுபவர்*

கையொப்பம் /

இடதுகை பெருவிரல் ரேகை

தேதி : *தேதி :*

ஆராய்ச்சி தகவல் தாள்

ஆய்வு தலைப்பு :

இடுப்பு குளியல் சிகிச்சையின் மூலம் சர்க்கரை நோயாளிகளின் இரத்த சர்க்கரை அளவில் சிகிச்சைக்கு முன் பின் ஏற்படக்கூடிய மாற்றத்தை ஆராய்ந்து அறியும் ஆய்வு

ஆய்வின் நோக்கம்:

இடுப்பு குளியல் சிகிச்சை எடுத்துகொள்ளும் சர்க்கரை நோயாளிகளின் இரத்த சர்க்கரை அளவில் சிகிச்சைக்கு முன் பின் ஏற்படக்கூடிய மாற்றத்தை கண்டறியவுள்ளோம் நீங்கள் .இந்த ஆய்வில் பங்கேற்று ஒத்துழைப்பு நல்கிட விரும்புகிறோம்.

இவ்வாய்வின்போது தங்கள் இரத்த சர்க்கரை அளவு முன் அருந்தும் உணவு) உணவு மற்றும்அருந்தியப்பின்னுமாக ஈடுபடுத்தும் ஆய்வில் ,இருமுறையும் வாரம் (இருமுறை இறுதியிலுமாக ஆய்வின் ,முன்பும்**FBS & PPBS** எனும் இரத்த பரிசோதனை செய்யப்படும் என்பதனை தெரிவித்துக்கொள்கிறோம்.

உங்கள் அனைத்து தகவல்களின் இரகசியம் பாதுகாக்கப்படும் .உங்கள் பெயரையோ அடையாளங்களையோ தங்கள் முன் அனுமதியில்லாமல் வெளியிடமாட்டோம் என உறுதியளிக்கின்றோம்.

இந்த ஆய்வில் பங்குக்கொள்வது தங்களின் தனிப்பட்டவிருப்பமாகும் .மேலும் நீங்கள் இந்த ஆராய்ச்சியில் இருந்து எந்த நேரமும் பின்வாங்கலாம் என்பதனையும் , இதனால் நீங்கள் பெரும் மருத்துவ சிகிச்சையில் எவ்வித குறைவோ அல்லது உங்களுக்கு தனிப்பட்ட முறையில் எவ்விதப்பாதிப்போ இருக்காது என்பதை தெரிவித்துக்கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம்

தேதி :

பங்கு பெறுபவர் / கையொப்பம்

இடதுகை பெருவிரல் ரேகை

தேதி :